doi: 10.1136/sti.2004.011726

Accepted for publication 2 June 2004

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Non-disclosure of previously known HIV seropositivity in patients ''newly'' diagnosed with HIV infection

We read with interest the letter from Natarajan *et al* regarding extensive unexpected antiretroviral resistance in an African immigrant patient.[1] The failure of HIV positive patients to disclose their status to healthcare workers has previously been documented with adverse clinical outcomes.[2]

Case reports

In this case series, we present five individuals who had previously been diagnosed with HIV, who then re-presented for HIV antibody testing and subsequent treatment without disclosing their HIV positive status. All cases were of African origin and diagnosed between October 2002 and February 2003.

Case 1

We were alerted to the possibility of a previously known HIV diagnosis in this woman as her mean corpuscular volume (MCV) was raised at 118 fl and she had features suggestive of the lipodystrophy syndrome. This patient finally revealed her previously known HIV diagnosis after a period of discussion with both physician and health adviser. She had extensive antiretroviral resistance and required optimisation of her antiretroviral treatment regimen.

Case 2

This patient revealed her previous diagnosis and antiretroviral treatment history after a period of discussion regarding treatment initiation. She realised that she may have antiretroviral resistance from previous suboptimal drug treatment.

Case 3

This patient revealed her known HIV diagnosis after a prolonged period of discussion. A decision to observe her immunological status was made in view of her apparently ''low'' viral load and reasonable CD4 count. She had not expected this decision and eventually ran out of drugs. She then revealed her previous history as she was becoming symptomatic and therefore keen to recommence therapy and. Her nadir CD4 count was <100 cells $\times 10^6$/l.

Case 4

Clinic staff at this centre recognised her from her previous attendances. In addition, her self reported demographics and signature from her previous attendance and most recent attendance matched completely. This patient subsequently transferred her care to a different HIV treatment centre where she subsequently revealed she had taken AZT while in Uganda but still insisted that she had never formally been tested.

Case 5

This patient was diagnosed in the antenatal clinic and was on antiretroviral therapy. She did not disclose this to us and alleged that she was given the medication by her husband for malaria.

Comment

Patients may fail to disclose their HIV diagnosis for a variety of reasons. These include fear of discrimination, fear that disclosure may jeopardise their asylum application and also concerns as to how they may be treated. In most cases the reasons are complex and involve many different factors.

Non-disclosure can result in numerous adverse outcomes for the individual.

Possible consequences for the patients include inappropriate clinical decisions owing to failure to recognise pre-existing antiretroviral drug resistance and toxicities, failure to recognise and address relevant social problems, the risk of inappropriate treatment when diagnosed antenatally, and the increased risk of mother to child transmission.

The number of programmes providing antiretroviral therapy in resource poor settings is increasing. Resistance to antiretroviral drugs in sub-Saharan Africa has been documented in several countries.[3]

Clinical clues to previous HIV diagnoses and antiretroviral drug exposure include haematological (raised MCV) and biochemical (raised lipids). Patients may also have morphological changes such as lipodystrophy and pigmentation. In addition, patients with an inappropriately low viral load and low CD4 count may have been previously thought to have a non-B clade viral subtype. This supposition may not always be accurate. Clinicians meeting such patients should look for other signs of antiretroviral drug exposure.

Therapeutic drug monitoring (TDM) and genotypic resistance testing may also be useful in selected cases. All five of these cases have undergone genotypic resistance testing, three of whom showed extensive multi-class resistance.

In all cases, disclosure occurred after multiple clinic attendances. It is highly probable that other cases of non-disclosure have occurred within this service. Clinicians should consider the possibility of HIV status non-disclosure and previous exposure to antiretrovirals when seeing ''newly diagnosed'' patients with HIV.

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Table 1 Details of cases with HIV*

	Case 1	Case 2	Case 3	Case 4	Case 5
Sex	F	F	F	F	F
Age	46	32	37	31	33
Ethnicity	White	Black African	Black African	Black African	Black African
Place of original HIV diagnosis	Congo	Kenya	Uganda	UK	Cameroon
Time from original HIV diagnosis to presentation for HIV testing	14 years	6 years	5 months	15 months	15 months
Route of HIV acquisition	Heterosexual sex	Heterosexual sex	Heterosexual sex	Heterosexual sex	Heterosexual sex
CD4 count at first presentation ($\times 10^6$/l)	354 (32%)	130(7%)	258(25%)	227 (12%)	294 (20%)
Viral load at first presentation (copies/ml)	1989	90 607	<50	424	<50
MCV at initial presentation (fl)	118		78.3	101.9	96.9
Exposure to antiretroviral therapy	• Multiple ARV exposure since 1989 outside UK • On failing regimen	• DDI • AZT+3TC • Last exposure 3 years ago	• Ran out of and discontinued d4T/3TC/EFV shortly after baseline bloods	• AZT monotherapy between initial and subsequent diagnosis	• DdI/d4T/HU May 00–Feb 01 • Trizivir Jul 02–Apr 03
Number of consultations before disclosure	5	4	6	9	3
Time to disclosure	8 weeks	8 weeks	6 weeks	16 months	4 months
Reason for non-disclosure	Believed that treatment would be denied	Concerned about disclosure to current partner of 3 years	Concerned about impact of knowledge of diagnosis on asylum application	Unknown	Thought she was being treated for malaria by husband

*See case reports.

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doi: 10.1136/sti.2004.011627

Accepted for publication 8 June 2004

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Atypical presentation of lobar nephronia in an adult co-infected with HIV and hepatitis C

Lobar nephronia or acute focal bacterial nephritis is an acute, non-suppurative, focal, renal infection.[1] It usually presents with fevers and flank pain. In the general population it is well described in children. We report an adult co-infected with HIV and hepatitis C, who presented with meningism and bilateral lobar nephronia.

Case report

A 37 year old man was admitted with a 4 day history of headaches, fevers, and vomiting with a 2 week background of dysuria. On presentation with a seroconversion illness 3 years previously he received combination antiretroviral therapy (ARV) for 9 months. Four months before the current admission ARV was re-introduced for symptomatic HIV infection. The most recent CD4 count was 250 cells$\times10^6$/l, and HIV viral load was 107 000 copies/ml. Hepatitis C infection had recently been diagnosed and the patient was receiving weekly interferon alfa. His symptoms began the day after the fifth injection. On examination he was pyrexial, temperature 39°C, had meningism and abdominal tenderness in both right upper quadrant and left iliac fossa. Investigations showed C reactive protein (CRP) 265 (normal = 0–4) IU/l, neutrophils 11×10^9/l, and normal urea and creatinine. Cranial computed tomography (CT) and cerebrospinal fluid analysis were normal. Urinanalysis showed protein++ and blood+; urine culture was negative. Blood cultures grew *Escherichia coli*, which was treated with cefuroxime. Abdominal CT scan showed multiple low attenuation solid lesions with peripheral enhancement in both kidneys (fig 1A). The patient's symptoms rapidly settled. He completed a 4 week course of oral cephadroxyl. As *E coli* was cultured from blood and a repeat scan after completion of treatment was normal (fig 1B) the renal CT appearances were ascribed to lobar nephronia.

The CT appearance of lobar nephronia is of either a single, or more uncommonly, multiple lesions in either one or both kidneys. The appearances are of either inflammatory (hypodense wedge-shaped) areas, or mass-like lesions.[2 3] A radiological differential diagnosis for single lesions includes intrarenal abscess, renal carcinoma, and simple cyst. For multiple lesions, it includes microabscesses, lymphoma, hamartomata, and metastases.

The clinical severity lies between that of pyelonephritis and renal abscess and it is important to differentiate lobar nephronia from these pathologies as management differs both in duration of antibiotics and the need for drainage of renal abscess. Histologically, the conditions differ. By contrast with the tissue necrosis and liquefaction seen in an abscess, in lobar nephronia there is localised hyperaemia, interstitial oedema, and leucocyte infiltration. These features are less severe and are diffuse in acute pyelonephritis.[2] *E coli* is the most common causative organism. Other pathogens include *Proteus mirabilis, Staphylococcus aureus, Klebsiella* spp, *Pseudomonas aeruginosa*, and enterococci. Antibiotics are given for up to 6 weeks and relapse may occur.

The majority of reports of lobar nephronia in the general population are in children, probably reflecting the higher incidence of urinary tract infections in children. Although lobar nephronia has been described previously in adult HIV infected patients,[4] our patient had an unusual presentation with meningism. Response to antibiotics was good and it is unclear to what extent immunosuppression due to HIV or hepatitis C infection, or interferon alfa may have contributed to the development of lobar nephronia. This case describes an uncommon presentation of renal infection in HIV infected adults and highlights the need to exclude differential diagnoses, especially lymphoma and metastases.

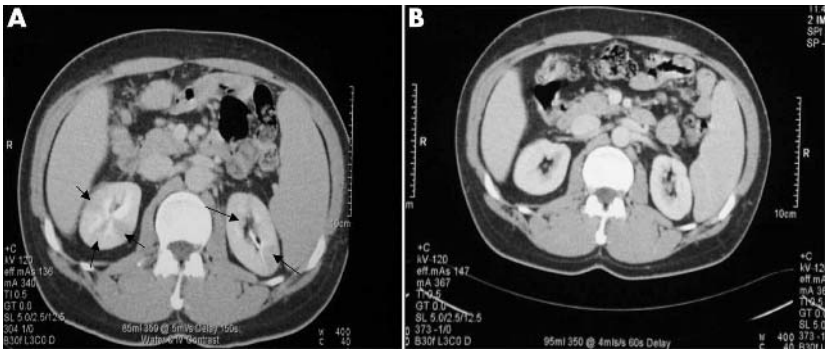

Figure 1 (A) CT scan of the abdomen showing multiple low attenuation solid lesions (arrows) with enhancing rims in both kidneys. There is associated splenomegaly, but no intra-abdominal lymphadenopathy and no ascites. (B) CT scan on completion of treatment. The appearance of the kidneys is normal.

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doi: 10.1136/sti.2004.011759

Accepted for publication 10 June 2004

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Molluscum contagiosum presenting as penile horn in an HIV positive patient

Dermatologists have the advantage of visualising the skin lesions and making the diagnosis. In immunocompetent patients most of the skin conditions have the characteristic clinical presentation and hence the diagnosis is made clinically by good visual impression. But the human immunodeficiency virus (HIV) has taken away this advantage. Owing to its profound effect on the immune system, the natural course and clinical features of most of the dermatological diseases have been altered. In this report we describe the unusual presentation of molluscum contagiosum as penile horn, in an HIV positive patient.

Case report

A 34 year old man presented with asymptomatic rapidly enlarging papular lesions on the penis and scrotum present for the past 6 months. He also had a significant weight loss and loss of appetite for the past month. On examination he was emaciated and had yellowish greasy scaling on the scalp, eyebrows, nasolabial folds, and chest. Examination of the lymphoreticular system did not reveal any abnormality. Genital examination revealed three well defined flesh coloured papules, two on the mucosal aspect on prepucial skin (one each at the 10 o'clock and 2 o'clock position) and the other one on scrotal skin near the root of the penis (fig 1). The size varied from 3 mm to 7 mm. All the lesions were non-tender and had keratotic projection in the centre, the height of which was more than its diameter. The scrotal lesion was fleshy and had a verrucous surface, and on pressing the lesion cheesy material could be expressed. Routine haemogram, liver, and renal function tests were within normal limits. Stool examination showed occasional *Cryptosporidium*. ELISA for HIV was positive. The CD4 count was just